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IN VIVO VOLATILE EMISSIONS FROM PEANUT PLANTS INDUCED BY SIMULTANEOUS FUNGAL INFECTION AND INSECT DAMAGE

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Abstract—Peanut plants, *Arachis hypogaea*, infected with white mold, *Sclerotium rolfsii*, emit a blend of organic compounds that differs both quantitatively and qualitatively from the blend emitted from plants damaged by beet armyworm (BAW; *Spodoptera exigua*) larvae or from uninfected, undamaged plants. Attack by BAW induced release of lipoxygenase products (hexenols, hexenals, and hexenyl esters), terpenoids, and indole. The plant-derived compound methyl salicylate and the fungal-derived compound 3-octanone were found only in headspace samples from white mold infected plants. White mold-infected plants exposed to BAW damage released all the volatiles emitted by healthy plants fed on by BAW in addition to those emitted in response to white mold infection alone. When BAW larvae were given a choice of feeding on leaves from healthy or white mold-infected plants, they consumed larger quantities of the leaves from infected plants. Exposure to commercially available (*Z*)-3 hexenyl acetate, linalool, and methyl salicylate, compounds emitted by white mold-infected plants, significantly reduced the growth of the white mold in solid-media cultures. Thus, emission of these compounds by infected plants may constitute a direct defense against this pathogen.

Key Words—Peanut, plant defense, volatile induction, white mold, beet armyworm, *Spodoptera exigua*, *Sclerotium rolfsii*, *Arachis hypogaea*.

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INTRODUCTION

Plants are attacked by a number of organisms during their life-span, including insect herbivores and disease-causing microorganisms. In response, they have evolved complex defense mechanisms to fend off their parasites. These mechanisms may be triggered by a specific organism and may be effective against present and future attacks by organisms of the same or different species. This phenomenon is known as cross-resistance and has been the subject of interest for many biologists since the 1970s (Karban and Baldwin, 1997). Another field that has received much attention, especially in the last decade, is the induction of what are now called indirect defenses (i.e., volatile emissions) in plants by feeding of herbivorous insects. Induced volatile compounds released by the plant attract natural enemies of the herbivore inflicting the damage (Dicke and Sabelis, 1988; Turlings et al., 1991).

It is clear that a thorough understanding of the plant's own defense mechanisms is required for their use against the pests that present a constant threat to our agricultural commodities. Although, in a typical field situation, plants are confronted with a wide array of antagonistic organisms, most studies on induced plant defenses have been conducted using plant–pathogen or plant–herbivore systems independent of one another. However, in recent years, many studies have suggested an interaction (or cross-talk) between the pathways involved in plant defense to pathogens and herbivorous insects. In many cases, it appears that induction of plant defenses by one type of organism interferes with plant defense against the other (Karban et al., 1987; Fidantsef et al., 1999; Stout et al., 1999; Bostock, 1999; Felton et al., 1999). Much remains to be learned about how plants defend against multiple stress factors. All studies conducted on this subject have focused on inducible direct defense compounds. It is still not known how plant indirect defenses against herbivores, consisting of volatile synomones, are influenced by the presence of phytopathogens, and how simultaneous attack of insects and phytopathogens on the plant may affect production of indirect defense substances.

In the present study, we tested the effect of peanut, *Arachis hypogaea* L. (Fabaceae), stem infection by the white mold fungus, *Sclerotium rolfsii* Sacc. (Mytosporic fungi), on the feeding preference of beet armyworm, *Spodoptera exigua* Hübner (Lepidoptera: Noctuidae). We also evaluated and compared the emission of volatile compounds from peanut plants under attack by the white mold fungus and by beet armyworm. The emission of volatile compounds from *S. rolfsii* fungal cultures was also evaluted. Finally, we tested the effect of synthetic forms of the commercially available compounds identified from the volatile profile emitted by fungus-infected plants on the radial growth of laboratory cultures of *S. rolfsii*.

METHODS AND MATERIALS

Fungal Culture. S. rolfsii (strain 80) was grown on potato dextrose agar (PDA) Petri plates from original cultures provided by Dr. Tim Benneman (Coastal Plain Experiment Station, University of Georgia, Tifton, Georgia). Subsequent cultures were started, under sterile conditions, in our laboratory by placing sclerotia harvested from the original culture in the center of the PDA media plates. Culture plates were kept in a biological incubator with a 14L:10D cycle and maintained at 25°C and 6–70% relative humidity. To inoculate the experimental plants, fungal culture plugs were cut out of the agar plate with a #2 cork borer (5 mm diam.).

Plant and Insect Material. Georgia Green peanut seeds were provided by Drs. Tim Brenneman and Glen Raines (Coastal Plain Experiment Station, University of Georgia, Tifton, Georgia). Seeds were sown in pairs in 3.78-liter pots (16 cm diam.) containing a 1:1 (v/v) mixture of commercially available filter sand and Metromix 300 (Scotts-Sierra Horticultural Company, Marysville, Ohio). Plants were grown in an insect-free greenhouse with natural light, under Florida summer conditions (14L:10D light cycle). The greenhouse temperature was kept at 25–30°C. After emergence, seedlings were thinned to one individual per pot. Each plant received 100 ml of a 3.38 g/liter liquid fertilizer solution [20-20-20 (N-P-K)]; Peters, W. R. Grace, Fogelsville, Pennsylvania) every two weeks starting one week after emergence. Five-week-old peanut plants with six fully developed leaves on the main stem and three fully developed leaves on each of two secondary branches were used in all experiments.

Beet armyworm eggs were obtained from the rearing facilities at the USDA-IBPMRL, Tifton, Georgia. Larvae were reared on a pinto-bean artificial diet following the methodology described by King and Leppla (1984). Insects were kept in a biological incubator with a 14L:10D cycle at 25°C. Third-instar larvae were used in all experiments.

BAW Feeding on S. rolfsii-Infected Peanuts. The feeding preference of BAW larvae on old and young leaves of healthy and fungus-damaged plants was evaluated to determine whether insects were deterred from feeding on tissues of the *S. rolfsii*-infected plants. Peanut plants were infected with the fungus by distributing four culture plugs along the first three internodal spaces of the main stem. The plugs were positioned so the fungus was in direct contact with the stem. Fungal plugs were pressed against the stem so they remained in place. Each plant was then individually covered with a 3.78-liter plastic storage bag (Ziploc DowBrands L. P., Indianapolis, Indiana) to provide adequate humidity and temperature conditions for fungal growth and colonization of the plant's stem. The plants were incubated for three days, after which time lesions approximately 1 cm long could be observed at the point of fungal contact with the stem. After this incubation period, bags were removed from the plants 24 hr before being used for the experiment. *S. rolfsii* is a

nonsystemic pathogen, and only the stems of the plants were in contact with the fungus; therefore, the leaves used for the experiments were not infected.

The second-oldest (old) or newest (young) fully developed tetrafoliate leaves on the mainstem of an infected plant were paired with their counterparts from a healthy plant by confining them within Petri dish clip-cages. The leaves (four leaflets each), still attached to each of the plants, were placed side by side within a clip-cage (Alborn et al., 1996), so the caterpillars had equal access to them. Insects were deprived of food for 6 hr before the start of the experiment to ensure immediate feeding on the plant tissues. Young peanut leaves are larger than old leaves, so six third-instar larvae were confined with the young leaves, and three larvae of the same stage were confined to the old leaves. Caterpillars were removed from the leaves after 24 hr. Leaves exposed to the feeding were carefully labeled, removed from the plants, brought into the laboratory, and photocopied to estimate feeding damage. Leaf images were scanned and imported into an imaging software program (ImagePC beta version 1, Scion Corporation, Frederick, Maryland) to estimate leaf area eaten and leaf area remaining. These measurements were used to calculate the leaf area consumed by the insects on each of the treatments. Six replicates of this experiment were set up at one time in the greenhouse. This experiment was repeated twice for a total of 18 replicates.

Volatile Collections from Fungus and BAW-Damaged Peanuts. In this experiment, plant treatments consisted of control (uninfected/undamaged), BAW damage, fungus-infection, and fungus-infection plus BAW damage. Plants were inoculated with the fungus as described in the first experiment. BAW-damaged plants were exposed to feeding by six third-instar larvae within the volatile collection chambers three days after pathogen inoculation and 12 hr before the start of the first sampling period.

The aerial portion of the plant was contained within glass sleeves that rested on Teflon bases, with an opening that closed around the plant stem (Röse et al., 1996). Purified air was pumped in at the top of the chamber at a rate of 5 liters/min. Air within each of the chambers was sampled daily, at a rate of 1 liter/min, for four days in three consecutive periods: (1) 6:00 AM–12:00 PM, (2) 12:00 PM–6:00 PM, and (3) 6:00 PM–6:00 AM. Compounds emitted were collected at the downwind end of the chambers in adsorbent traps containing 25 mg Super Q (800–100 mesh) (Alltech, Deerfield, Illinois). All volatile collections were conducted in the greenhouse where plants were grown. The experiment was set up in duplicate and repeated three times on different days for a total of six replicates.

Volatile Collection from Fungal Cultures. To determine whether any of the volatile compounds collected from infected plants were also produced by the fungus directly, we collected and analyzed volatiles from culture plugs of the white mold at different stages of development. Five 1-cm-diam. plugs were made from *S. rolfsii* culture plates at 3, 5, 8, and 16 days after inoculation. Volatiles emitted from control, noninoculated PDA plates, were also collected and analyzed. Plugs

from each treatment were separately placed in Pyrex glass odor collection chambers, and volatiles were collected using a push–pull system previously described by Turlings et al. (1991). Volatiles were adsorbed by the Super Q traps described previously. Approximately 300 ml/min of purified and humidified air was passed over the culture plugs and through the collection traps for a 2-hr period. Air samples were obtained from plugs made from three different culture plates for each of the developmental times tested.

Sample Extraction and Analysis. Compounds from individual traps in the volatile collection experiments were eluted with 170 μl dichloromethane (GC/GC-MS Solvent, B&J, AlliedSignal, Inc), and then 400 ng each of *n*-octane and nonyl acetate were added to each eluted sample as internal standards. The samples were analyzed by gas chromatography with flame ionization detection (HP5890 gas chromatograph, HP7673 auto sampler, Hewlett Packard, Palo Alto, California) equipped with a 15-m $\times$ 0.25-mm-ID, 0.25-μm film thickness DB-1 capillary column (Quadrex, New Haven, Connecticut). The splitless mode injector system was set at 220°C, the column oven was held at 40°C for 1 min after injection and then programmed at 14°C/min to 180°C. The carrier gas used was helium at a flow average velocity of 19 cm/sec. For identification of compounds, selected samples were analyzed via GC-MS (HP 6890 gas chromatograph equipped with 30-m $\times$ 0.25-mm-ID, 0.25-μm film thickness HP-5 capillary column, interfaced to a 5973 Mass Selective Detector, Hewlett Packard, Palo Alto, California) in both electron impact and chemical ionization modes. The column was held at 40°C for 1 min after injection and then programmed at 10°C/min to 180°C. The carrier gas used was helium at a flow average velocity of 30 cm/sec. Isobutane gas was used as the reagent gas for chemical ionization, and the ion source temperature was set at 250°C. Individual compounds were identified by comparing their retention times to that of commercially obtained authentic samples and by comparing their mass spectra against those available in a database from the Environmental Protection Agency/National Institute of Standards and Technology.

Effect of Volatile Compounds from S. rolfsii-Infected Peanut on Radial Growth of Fungal Cultures. The effect of vapors from synthetic forms of 3-octanone, (*Z*)-3-hexenyl acetate, linalool, and methyl salicylate (Sigma-Aldrich Chemical Company, St. Louis, Missouri) on the radial growth of *S. rolfsii* on PDA culture plates was evaluated. These compounds were selected because they are present in the blend of compounds emitted by infected peanut plants and because they were commercially available. Three levels of the compounds, 3, 10, and 30 μl, were diluted in 100 μl hexane (GC/GC-MS Solvent, B&J, AlliedSignal, Inc) and loaded on to 5 $\times$ 11-mm sleeve stoppers made of natural red rubber (Wheaton, Millville, New Jersey). The loaded stoppers were allowed to equilibrate for 24 hr before using them for the experiments. The rubber stoppers were attached to the cover of the Petri dish by means of double-sided Scotch tape to prevent direct contact and contamination of the growth media. Two *S. rolfsii* sclerotia were placed in the

center of the bottom part of the plate containing the PDA media; thus, the fungus was exposed only to the vapors of the compounds emitted from the rubber stoppers for the duration of the experiment. Rubber stoppers loaded with 100 μl hexane were used in the control plates. This experiment was repeated on different days for a total of six replicates.

Statistical Analyses. Data for BAW feeding preference were analyzed by paired *t* test (Proc MEANS; SAS Institute, 1996). Data for volatile collections and the effect of volatiles on fungal radial growth were analyzed using ANOVA (Proc GLM; SAS Institute, 1996). Significant ANOVAs were followed by Tukey's mean separation test.

RESULTS

BAW Feeding on S. rolfsii-Infected Peanuts. BAW feeding was not negatively affected by infection of *S. rolfsii* on peanut plants. To the contrary, larvae consumed significantly more of the young and old leaves from fungus-infected plants than from healthy, noninfected plants (Figure 1). Fungal infection resulted in a 1.4- and 6.4-fold increase in BAW leaf area consumption for old and young leaves, respectively.

Volatile Collections from Fungi and BAW-Damaged Peanuts. Uninfected/uninfested control plants released small amounts of volatiles compared to the other treatments. Healthy and fungus-infected peanut plants exposed to BAW feeding started releasing volatiles within 24 hr. However, plants exposed to damage by *S. rolfsii* alone did not emit volatiles until the third day of collection. Volatile emission in all plants showed a diurnal pattern with peak release during period 2 (12:00–6:00 PM). Thus, data presented for this experiment are from 12:00–6:00 PM of the third day of collection.

Noninoculated control plants (Figure 2A) released relatively small amounts of volatiles, such as myrcene, β-ocimene, linalool, and (*E*)-4,8 dimethyl-1,3,7-nonatriene compared to those released by white mold-infected or BAW-damaged peanut plants. Plants infected with the white mold fungus alone released (*Z*)-3-hexenyl acetate, linalool, and relatively large amounts of (*E*)-4,8-dimethyl-1,3,7-nonatriene (Figure 2B), which were also present in emissions from BAW-damaged plants. Additionally, the compounds methyl salicylate and 3-octanone were only present in volatile emissions from plants that were infected by the fungus (Figure 2B) and not in those of plants damaged by BAW alone (Figure 2C). Noninfected peanut plants exposed to feeding by BAW released large amounts of lipoxygenase products, monoterpenes, indole, and sesquiterpenes (Figure 2C). The emission of volatiles from peanuts in response to BAW damage was not negatively affected by infection of white mold fungus on the plant. In fact, white mold-infected peanuts damaged by BAW (Figure 2D) released all the volatiles

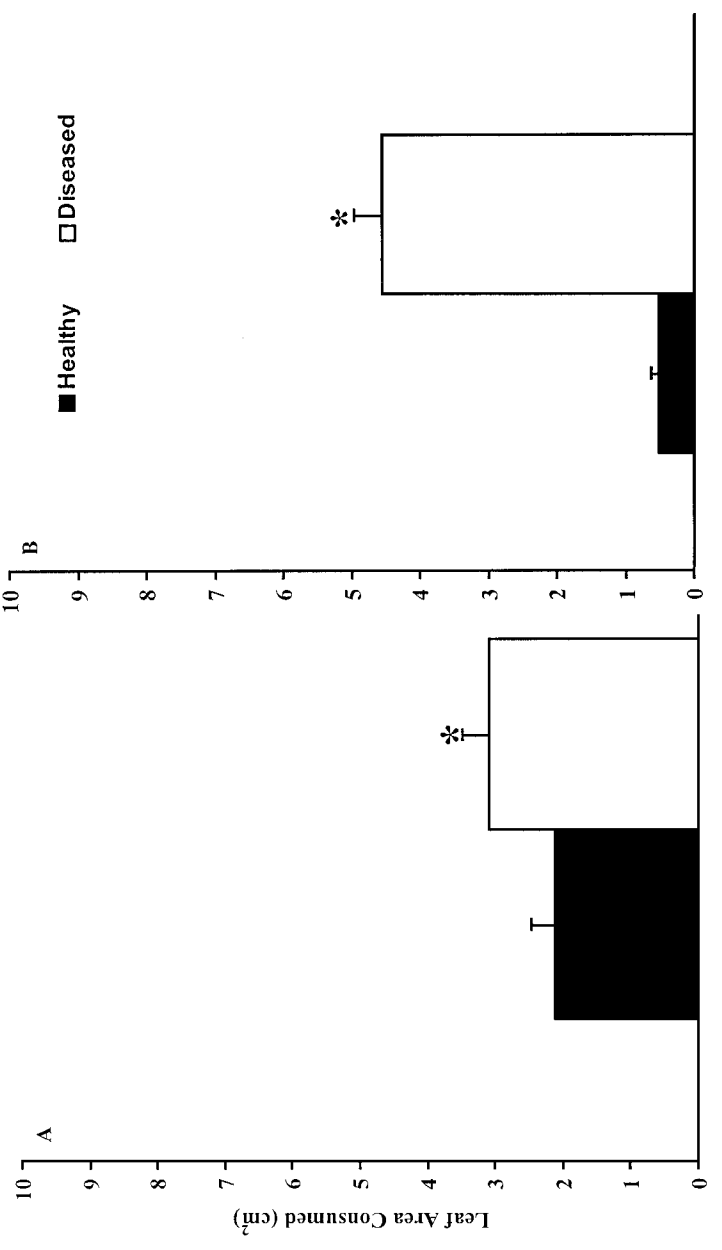

FIG. 1. Mean leaf area consumed by third-instar *Spodoptera exigua* larvae in paired-choice tests with healthy (dark bars) and white mold-infected (white bars) plants. (A) Old leaves and (B) young leaves. Error bars denote 1 SD and an asterisk denotes significant differences (paired t test, $P \leq 0.05$).

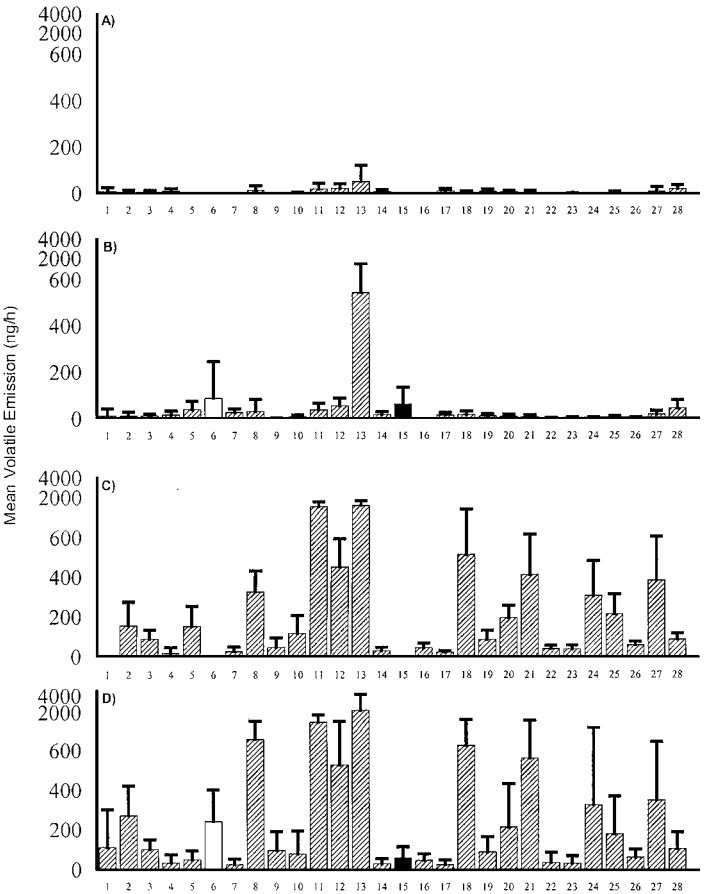

FIG. 2. Mean volatile emissions from peanut plants: (A) uninfected/undamaged, (B) white mold-infected, (C) BAW-damaged, and (D) white mold-infected + BAW-damaged. Compounds: (1) (*E*)-2-hexenal, (2) (*Z*)-3-hexen-1-ol, (3) α-pinene, (4) β-pinene, (5) 1-octen-3-ol, (6) 3-octanone, (7) (*Z*)-3-hexenyl acetate, (8) myrcene, (9) eucalyptol, (10) limonene, (11) β-ocimene, (12) linalool, (13) (*E*)-4,8-dimethyl-1,3,7-nonatriene, (14) (*Z*)-3-hexenyl isobutyrate, (15) methyl salicylate, (16) (*E*)-2-hexenyl butyrate, (17) (*Z*)-3-hexenyl butyrate, (18) indole, (19) *cis*-jasmone, (20) β-caryophyllene, (21) 2,6,-dimethyl-6-(4-methyl-3-pentenyl) bicyclo[3.1.1]hept-2-ene, (22) α-farnesene, (23) α-humulene, (24) germacrene-D, (25) β-farnesene, (26) naphthalene, (27) nerolidol, (28) (*E*,*E*)-4,8,12-trimethyl-1,3,7,11-tridecatetraene. Day 3, period 2 (12:00–6:00 PM). Bars across treatments headed with the same letter are not significantly different (Tukey's mean separation test, $P \geq 0.05$). Error bars are equivalent to 1 SD. No synthetic standards were available for compounds 21, 24, and 26; identification is based on National Institute of Standards and Technology library spectral match only.

typical of a healthy plant damaged by BAW. Furthermore, the amounts of some released compounds, such as (*E*)-2-hexenal, (*Z*)-3-hexen-1-ol, myrcene, and (*E*)-4,8-dimethyl-1,3,7-nonatriene, were higher than those emitted from noninfected plants in response to BAW feeding (Figure 2C). In addition, headspace collections from white mold/BAW-damaged plants also contained volatiles produced by plants in response to white mold infection alone (Figure 2B).

Volatile Collection from Fungal Cultures. Volatile profiles from *S. rolfsii* cultures showed the presence of one dominating volatile compound, which was identified as 3-octanone. The amounts of 3-octanone produced by the fungal culture plugs increased with age up until day 8, and ceased almost completely by day 16. Amounts of 3-octanone produced were 0.9 $\pm$ 0.4 ng/hr (mean $\pm$ SE), 1.8 $\pm$ 0.3 ng/hr, 4.3 $\pm$ 0.7 ng/hr, and 0.1 $\pm$ 0.2 ng/hr at days 3, 5, 8, and 16, respectively. This compound was recovered only from culture plates containing the fungus and not from non inoculated control PDA plates.

Effect of Selected Volatile Compounds from S. rolfsii-Infected Peanut on Radial Growth of Fungal Cultures. The growth rate of the fungus was significantly reduced by (*Z*)-3-hexenyl acetate, linalool, and methyl salicylate volatiles released from septa treated with 10 μl. Additionally, volatiles from 30 μl of linalool or methyl salicylate on a septum completely inhibited sclerotial germination and fungal growth (Figure 3). The *S. rolfsii*-produced compound 3-octanone did not prevent sclerotial germination and significantly reduced the growth of the fungus only at the highest dose of 30 μl tested.

DISCUSSION

Plant release of volatile compounds in response to attack by herbivores and the role of such compounds in attracting parasitoids of the herbivores have been studied extensively in recent years (Turlings et al., 1991, 1993; McCall et al., 1994; Loughrin et al., 1995; Röse et al., 1996, 1998; Paré and Tumlinson, 1997). In contrast, the emission of volatiles in response to pathogen invasion has been the subject of a limited number of studies (Croft et al., 1993; Doughty et al., 1996; Shualev et al., 1997; Chaudry et al., 1998; Buonaurio and Servili, 1999). All of these studies have examined the induction of plant volatile emission by either herbivore damage or pathogen infection on different plant systems or using excised plant parts. Thus, we have no clear knowledge of whether the regulation of volatile production in response to these organisms is affected in any way by the simultaneous attack of herbivores and phytopathogens on the same plant.

Data obtained from this study confirm that peanut plants release volatile compounds in response to attack by the white mold fungus. Furthermore, we present conclusive evidence that the volatile profile emitted by these plants differs qualitatively and quantitatively from profiles emitted from healthy plants and from

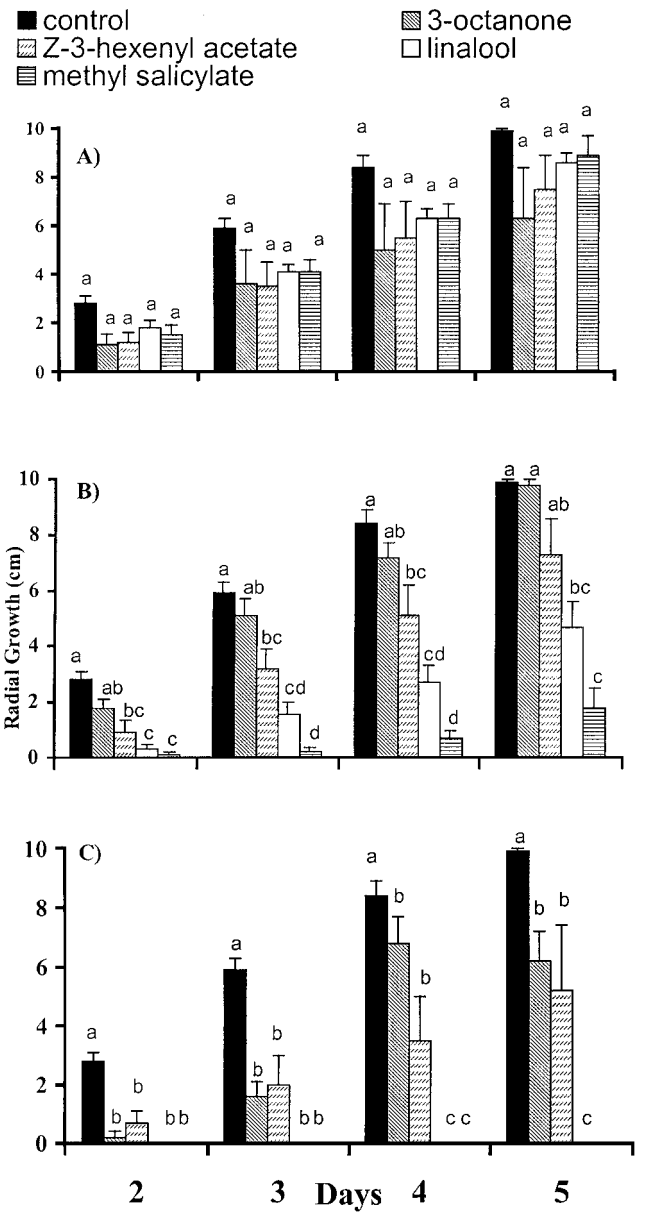

FIG. 3. Mean radial growth of *S. rolfsii* cultures exposed to volatile vapors from 3-octanone, (*Z*)-3-hexenyl acetate, linalool, and methyl salicylate: (A) 3 μl, (B) 10 μl, and (C) 30 μl of the synthetic forms of the compounds. Bars within days headed with the same letter are not significantly different (Tukey's mean separation test, $P \geq 0.05$). Error bars indicate 1 SD.

those emitted in response to BAW damage. Additionally, previous infection of the plant by the white mold fungus does not interfere with the emission of volatiles by the diseased plant in response to BAW attack; rather it seems to induce release of some compounds in relatively higher quantities.

The significant quantitative difference in the volatile profile of peanut plants attacked by either the fungus or the insect, added to the fact that emission of compounds was not suppressed when both organisms were simultaneously attacking the plant, provides a clear indication that the activation and regulation of plant biosynthetic pathways is dependent upon the type of threat perceived by the plant. Four major biosynthetic pathways are believed to be involved in the production of plant volatiles in response to damage by lepidopterous larvae (Paré and Tumlinson, 1997). The shikimic/tryptophan, mevalonate, Rohmer, and lipoxygenase pathways are all believed to be involved in the production of the different classes of volatile compounds released by plants under attack by insect herbivores (Paré and Tumlinson, 1997). The lipoxygenase (LOX) pathway, via jasmonate production, has been directly linked to direct plant defense responses to wounding and damage by herbivorous insects (Ryan, 1990; Farmer and Ryan, 1990; Farmer et al., 1992). Jasmonic acid and methyl jasmonate have also been found to induce volatile emissions similar to those resulting from herbivory in many plants (Boland et al., 1995; Dicke et al., 1999). Thus, it has been suggested that they mediate volatile production. In our study, the emission of volatiles from peanut plants previously infected with *S. rolfsii* indicates that, in this case, unlike direct defenses, volatile production is not compromised by pathogen infection. The latter may be an indication that peanut production of volatile compounds in response to insect and pathogen attack is not jasmonate-dependent. However, the precise combination and regulation of pathways activated in response to an individual or combination of stressors may vary widely among plant species. Therefore, the role of jasmonic acid and salicylic acid on the production and release of induced volatiles by plants in response to insect and pathogen attack needs to be investigated further. The dynamics of volatile emission in response to insect herbivores, phytopathogens, and their combined effect upon other plant species also merit additional attention.

Volatile compounds have been shown to affect pathogens in different ways. For example, the germination and growth of white mold are stimulated by the release of methanol and other volatile compounds emanating from moist peanut hay (Shokes et al., 1996). On the other hand, volatiles from ground-up healthy corn kernels resistant to *Aspergillus flavus* have been found to inhibit the growth and aflatoxin production in colonies of this pathogen (Zeringue et al., 1996). In cotton, however, the lipoxygenase-derived volatile (*E*)-2-hexenal inhibited, while α- and β-pinene stimulated the growth of this fungus (Zeringue and McCormick, 1989, 1990). Thus, the negative effect of (*Z*)-3-hexenyl acetate, linalool, and methyl salicylate volatiles on the growth of our *S. rolfsii* in laboratory cultures suggests that the production of these volatiles by the plant acts as a direct defense by

slowing the growth of the fungus and preventing additional sclerotial germination. However, since we were unable to quantify the headspace concentration of the compounds used, further experiments need to be conducted to confirm that the amounts are comparable to those produced by an infected plant and that in fact the growth of the fungus is hindered under natural conditions.

In the dual-choice feeding experiments, BAW preferentially fed on leaves from white mold-infected peanut plants. BAW and corn earworm, *Helicoverpa zea*, have been reported to feed more upon tomato plants that had been previously treated with benzothiadiazole–carbothionic acid *S*-methyl ester (BTH), an elicitor of systemic acquired resistance via the salicylic acid pathway (Stout et al., 1999). Tomato plants treated with BTH were found to have compromised direct defenses, based on expression of genes encoding for proteinase inhibitors (Fidantsef et al., 1999). In our study, the presence of methyl salicylate in headspace samples of white mold-infected plants indicates activity of the salicylic acid pathway in peanuts in response to *S. rolfsii* infection. Thus, the feeding preference observed in BAW towards leaves from white mold-infected peanut plants may be caused by a reduction in direct defenses of the plant due to fungal infection. Alternatively, the feeding preference of BAW for leaves from fungus-infected plants could be due to changes in the nutritional quality of the tissues caused by the pathogen infection. Different pathogen species have been found to either up- or down-regulate accumulation of photoassimilates (reviewed in Hatcher, 1995). Pathogen infections influence concentrations of compounds such as nitrogen, nonstructural carbohydrates, starch, protein, free amino acids, and amino acid composition, in their respective plant hosts (reviewed in Hatcher, 1995). Although most of the studies included in that review evaluated the effect of leaf-infecting pathogens on the suitability of plant tissue to insect herbivores, it is not inconceivable that a stem-infecting pathogen such as *S. rolfsii* can have similar consequences upon its hosts. However, this is an aspect that remains to be elucidated.

To our knowledge, this is the first in planta study in which the production of volatiles by a single host system in response to both insect herbivores and pathogens has been evaluated simultaneously. This is also the first time that the effect of previous pathogen infection on the production of plant volatiles in response to insect damage has been studied. The study of plant volatile defenses may improve our understanding of plant resistance mechanisms to disease and insect herbivores. The identification of specific pathogen- and herbivore-induced plant volatiles will greatly contribute to the development, improvement, and implementation of host-plant resistance and other control methods for insect and pathogen pests.

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